Subdural Hemorrhage in Patients with End-Stage Renal Disease Requiring Dialysis: A Single-Center Study

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Abstract

Purpose: Patients with end-stage renal disease (ESRD) have higher risks of subdural hemorrhage (SDH) and subsequent 30-day mortality. However, evidences regarding optimal mode of dialysis therapy during acute management are sparse. We aimed to compare the outcomes of ESRD patients who received continuous peritoneal dialysis (CPD) or extended hemodialysis (EHD) after SDH and determined factors associated with 30-day mortality.

Methods: We retrospectively reviewed consecutive patients with SDH and ESRD in a medical center. The clinical parameters and outcomes were compared between CPD and EHD groups. Factors associated with 30-day mortality were analyzed.

Results: We reviewed 32 patients, including 22 received EHD, 8 received CPD, and 2 received continuous veno-venous hemodialysis. Neurosurgery was done in 9 (28%) of them. There was no significant difference in baseline parameters and outcomes between EHD and CPD groups. The overall 30-day mortality rate was 19%. Lower Glasgow coma scale (GCS, median [interquartile range]: 10 [7-12] vs. 15 [11-15], $p = 0.02$) and larger changes in absolute mean arterial pressure (MAP: 26.5 [10.5-46.0] vs. 7.5 [2.0-17.8] mmHg, $p = 0.01$) during the first dialysis therapy were noted in patients with 30-day mortality. In multivariate analysis, consciousness disturbance at presentation was an independent risk factor for 30-day mortality.

Conclusion: Among ESRD patients with SDH, the 30-day mortality rates were similar between EHD and CPD groups. MAP change during dialysis might be an important modifiable risk factor for 30-day mortality, though the effect was not significant in multivariate analysis. Further prospective studies with larger sample size are warranted.

Keywords: subdural hemorrhage; end-stage renal disease; peritoneal dialysis; hemodialysis; outcome.

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INTRODUCTION

The incidence and prevalence rates of treated end-stage renal disease (ESRD) in Taiwan are 455 and 3219 per million population/year, respectively, which are amongst the highest in the world[1]. Due to coagulation abnormality and platelet dysfunction, patients with ESRD may have higher risks of bleeding events, e.g., intracranial bleeding[2,3]. For example, the incidence of subdural hemorrhage (SDH) in Taiwanese patients with ESRD requiring hemodialysis is 4.47-fold higher than control, while the adjusted hazard ratio (HR) is 3.81[4]. Given the high prevalence of ESRD in Taiwan and the associated higher risk of SDH, it may be anticipated that we neurologists or neurosurgeons will encounter more and more ESRD patients presenting with SDH requiring emergent treatment.

A recent nationwide study reports that the 30-day mortality of SDH in Taiwanese patients with ESRD is as high as 35.2%, compared with 8.5% in the control group[4]. The higher comorbidity burden and the more utilization of oral antithrombotic agents at baseline may contribute to the higher mortality risk in ESRD patients with SDH[4]. And aside from the increased bleeding tendency, treatment of SDH in ESRD patients may be further complicated by unstable hemodynamic parameters and increased intracranial pressure (ICP) during dialysis[5]. However, evidences regarding the treatment, including dialysis therapy in ESRD patients with SDH, have still been sparse[5].

Although continuous modalities of renal replacement therapy provide an advantage for the patients with compromised cerebral perfusion[6], they are generally limited to the intensive care units setting. Many hemodialysis (HD) patients with SDH are managed in the general wards. In such condition, these patients are treated by intermittent HD, which can potentially exacerbate brain injury by both causing an increase in intracranial pressure (ICP)[7,8], and excessive ultrafiltration[9] leading to reduced cerebral perfusion[10]. Thus, the dialysis prescription should be altered to minimized changes in serum osmolality and drop in blood pressure during HD[5]. Tietjen et al described a case of SDH with onset during hemodialysis, after which the patient was successfully managed with temporary peritoneal dialysis (PD)[11]. Changing the mode of dialysis from HD to PD in patients with SDH may be beneficial in improving overall survival[12]; however, this experience has rarely been reported.

In our hospital, we tried to provide dialysis therapy with a stable hemodynamic state for our ESRD patients with acute SDH by using continuous peritoneal dialysis (CPD), extended hemodialysis (EHD) with a modified prescription (low dialysate flow, low blood flow and heparin-free with intermittent saline flush), or continuous veno-venous hemodiafiltration (CVVHD). In the present study, we report our single-center experience of management of patients with SDH and ESRD requiring dialysis, compare the outcomes of patients receiving CPD or EHD after the onset of SDH, and identify factors potentially associated with the prognosis.

METHODS

Study Cohort

The institutional review board of National Cheng Kung University Hospital approved the study protocol (IRB NO.: A-ER-106-047). We retrospectively searched consecutive hospitalized patients with discharge diagnoses of both SDH and ESRD from January 1, 2005 through Mar 31, 2010. The diagnosis of SDH was made by computed tomography (CT) scan of the brain. The medical records and radiological images were reviewed. We excluded patients who received no dialysis treatment during the hospitalization for SDH, who were not SDH cases after image re-evaluation, or who had incomplete clinical data. Finally, thirty-two patients were included in subsequent analysis (Figure 1).

Managements

Patients would shift to EHD, CPD, or CVVHD after hospitalization for SDH. Different model of dialysis therapy was chosen for an individual patient, according to the constitutional condition of the patient (e.g. not suitable for PD due to previous history of abdominal surgery), the will of the patient or surrogate, and upon the decisions of the consulting nephrologists and the neurocritical care team members. Once CPD had been decided, we would immediately consult the general surgeon to place the temporary PD catheter. The procedure of PD catheter insertion has been described previously[8]. In brief, PD

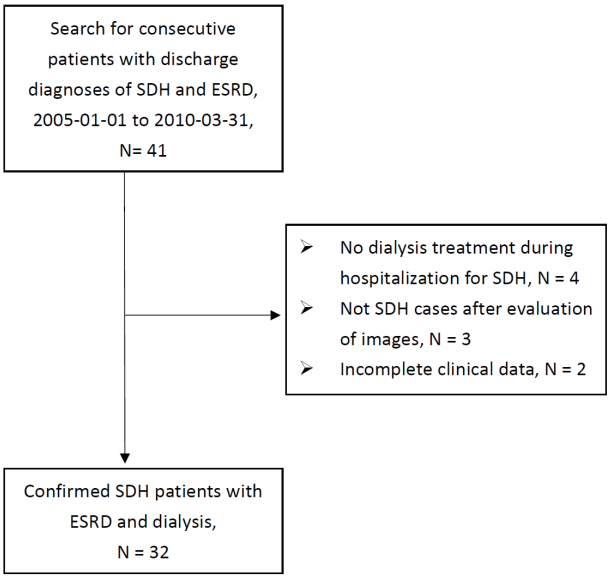

Figure.1 Patient selection flowchart. Abbreviations: SDH: subdural hemorrhage; ESRD: end-stage renal disease.

catheter was placed approximately 2 cm below the umbilicus and gently advanced into the peritoneal cavity. A purse-string suture was used to secure the catheter into place. All of the CPD patients were started on low-volume (0.5 liter) exchanges and gradually increased to adequate fill volume (1.5-2 liter per exchange) if no leakage of dialysate was found. The EHD protocol was low dialysate flow: 300 ml/min; low blood flow: 150 ml/min; heparin-free dialysis with intermittent saline flushes; longer duration: 4–6 h or more; frequency: three times a week. The amount of fluid removal was carefully evaluated at each dialysis session by nephrologist or neurosurgeon to avoid significant blood pressure change during the dialysis session. The protocol for CPD was continuous ambulatory PD or continuous cyclic PD. The PD nurse would assist the CPD program. We resumed ordinary standard HD after the consensus of the care team that the patients became neurologically stable.

After hospitalization, all patients underwent the same protocol to ICP, such as elevating head of bed to 30-45 degree , avoid hypotension, hypoxia, and fever, normocarbia ($PaCO_2$ = 35-40 mmHg), keep cerebral perfusion pressure > 60 mmHg (in patients with ICP monitor placement), etc. The surgical indications included persistent elevation of ICP and neurologic deficits arising from the mass effect by the hematoma. The neurosurgical procedures included procedures of unilateral or bilateral burr hole drainage, craniotomy, and craniectomy.

Variates and Outcomes

We retrieved the baseline demographics, underlying medical history and prior medication, clinical presentation, laboratory data, radiological findings available at the initial evaluation of SDH, and modes of dialysis among the study cohort. The CT scan was retrospectively evaluated by a single investigator (Jung-Shun Lee) who was a board-certified neurosurgeon and blind to the outcomes of patients. The SDH was classified as acute, subacute, and chronic, while the length of hematoma (in millimeter) was measured as the thickness of the SDH. We further explored details of neurosurgery and initial dialysis therapy after hospitalization, including changes of mean arterial pressure (MAP) between the beginning and the end of the first session of EHD, or that between the beginning and the end of the first day of CPD. We also recorded relevant management for abnormal MAP.

Our primary outcome was mortality at the 30 days after hospitalization. We also recorded rates of major complications during hospitalization as secondary outcomes, including enlarge of hematoma and dialysis-related complications.

Statistical analysis

Results were presented as numbers and percentages for categorical variables, as mean ± standard deviation (SD) for normally distributed continuous variables, and as median and interquartile range for ordinal or non-normally distributed continuous variables. Baseline demographics and initial clinical variates were compared between CPD/EHD groups and also between patients with and without 30-day mortality. Ordinal or continuous variables were compared with Mann-Whitney test, and categorical variables were compared with Chi-square test or Fisher's exact test, as appropriate. Univariate regression analysis was done to explore the unadjusted effect of the selected variable on 30-day mortality. Each variable with a *p*-value less than 0.05 in univariate analyses was included in the multivariate binary logistic regression (forward stepwise) analysis. A two-tailed *p*-value < 0.05 was considered

statistical significant.

RESULTS

Among the 32 patients we analyzed, the age was 68 ± 10 years, 66% of them were male, and all but one patients received chronic HD before hospitalization for SDH. The median duration of dialysis before onset of ICH was 24 months (interquartile range, 9.5-61 months). Other baseline demographics of the patients were listed in Table 1. Six (19%) patients died within 30 days.

The only one previously non-dialyzed patient received EHD for uremia developing after SDH. And among the 31 chronic HD patients, 8 patients received CPD, 2 patients received CVVH, and the other 22 patients received EHD after SDH. The comparison of baseline demographics, clinical characteristics, radiological characteristics, first post-SDH dialysis parameters, neurosurgery and outcomes were presented in Table 2 (for the purpose of the present study, two patients received CVVHD were not included in analysis here). The CPD group was younger, had higher percentage of diabetes, when compared with the EHD group. Other baseline demographics, clinical characteristics, and radiological

Table 1. Baseline demographics of patients at onset of SDH (total N=32)

Age, years	68 ± 10
Male	21 (66)
Hypertension	24 (75)
Diabetes	16 (50)
Old stroke	9 (28)
Coronary artery disease	3 (9)
Atrial fibrillation	4 (13)
Prior use of aspirin or warfarin	7 (22)
Dialysis duration before SDH, months[1]	24 (9.5-61)

Abbreviation: SDH, Subdural hemorrhage.

Data are presented as mean ± SD (normally distributed data), median (interquartile range) (non-normally distributed data), or number (percentage).

[1]One patient had end-stage renal disease without hemodialysis before onset of SDH

Table 2. Demographics, clinical and radiological characteristics, first post-SDH parameters and outcome for CPD and EHD groups

	CPD (n = 8)	EHD (n = 22)	*p* value
Baseline Demographics			
Age, years	61 ± 8	71 ± 10	0.02
Male	6 (75)	14 (64)	0.68
Hypertension	7 (86)	15 (68)	0.39
Diabetes	7 (88)	8 (36)	0.04
Coronary artery disease	1 (13)	2 (9)	1.00
Dialysis duration, months	30.0 (10.5- 114.0)	23.5 (5.5-62)	0.57
Clinical characteristics			
Known head injury before SDH	6 (75)	17 (77)	1.00
Time from symptoms onset to presentation, days	0.3 (0.1-2.8)	0.5 (0.1-4.0)	0.59
Consciousness disturbance	5 (63)	6 (27)	0.10

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Table 2. (Continued) Demographics, clinical and radiological characteristics, first post-SDH parameters and outcome for CPD and EHD groups

	CPD (n = 8)	EHD (n = 22)	*p* value
Cranial nerve palsy	0 (0)	2 (9)	1.00
Focal weakness	0 (0)	6 (27)	0.16
Seizure or convulsion	0 (0)	2 (9)	1.0
Initial Glasgow coma scale	11 (6-15)	15 (12-15)	0.19
Radiological characteristics			
Acute SDH	5 (63)	10 (50)	0.69
Bilateral SDH	2 (25)	1 (5)	0.17
Skull fracture	2 (25)	4 (19)	1.00
Thickness of SDH, mm	13.3 ± 6.2	9.2 ± 4.5	0.18
First post-SDH dialysis parameters			
Duration between SDH onset and first dialysis, days	3.5 (2.3-4.8)	4.0 (2-6.3)	0.77
MAP change during first dialysis (mmHg)	7 (2.8-25.0)	12 (2.8-21.5)	0.77
Labetalol or CCB treatment during first dialysis	4 (50)	4 (18)	0.16
Neurosurgery	4 (50)	4 (18)	0.16
Outcomes			
Enlargement of hematoma	1 (12.5)	2 (9)	1.00
30-d mortality	2 (25)	3 (14)	0.59

Abbreviation: SDH, subdural hemorrhage; GCS, Glasgow coma scale; MAP, Mean arterial pressure; CCB, calcium channel blocker. Data are presented as mean ± SD (normally distributed data), median (interquartile range) (non-normally distributed data), or number (percentage). MAP changes between the start and end of the first EHD, and of the first day of CPD. Two patients received CVVHD were not included for comparison.

Table 3. Comparison of baseline demographics, clinical and radiological characteristics, and hospitalization courses between SDH patients with and without 30-day mortality (total N = 32)

	With 30-day mortality (n = 6)	Without 30-day mortality (n = 26)	*p* value
Baseline Demographics			
Age, years	70 ± 8	68 ± 11	0.83
Male	6 (100)	15 (58)	0.07
Hypertension	5 (83)	19 (73)	1.00
Diabetes	3 (50)	13 (50)	1.00
Old stroke	9 (35)	0 (0)	0.15
Coronary artery disease	0 (0)	3 (12)	1.00
Atrial fibrillation	0 (0)	4 (15)	0.57
Prior use of aspirin or warfarin	0 (0)	7 (27)	0.30
Dialysis duration before SDH, months	12.0 (7.8-159.0)	29.5 (9.8-61)	0.83
Clinical characteristics			
Known head injury before SDH	5 (83)	20 (77)	1.00
Time from symptoms onset to presentation, days	2.4 (0.4-8)	0.3 (0.1-2.4)	0.05

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Table 3. (Continued) Comparison of baseline demographics, clinical and radiological characteristics, and hospitalization courses between SDH patients with and without 30-day mortality (total N = 32)

	With 30-day mortality (n = 6)	Without 30-day mortality (n = 26)	*p* value
Consciousness disturbance	5 (83)	7 (27)	0.02
Cranial nerve palsy	1 (17)	1 (4)	0.35
Focal weakness	2 (33)	5 (19)	0.59
Seizure or convulsion	1 (17)	1 (4)	0.35
Initial Glasgow coma scale	11 (9-15)	15 (11-15)	0.16
Radiological characteristics			
Acute SDH	2 (33)	15 (58)	0.27
Bilateral SDH	0 (0)	3(12)	1.0
Skull fracture	2 (40)	4 (15)	0.24
Thickness of SDH, mm	12 ± 5	10 ± 5	0.45
Laboratory data			
Hb, gm/dl	10 ± 1	11 ±1	0.33
Platelet, 10^3/cmm	109 (93-235)	162 (128-220)	0.26
PT, seconds	12 (11-14)	11 (11-12)	0.23
APTT, seconds	32 (25-61)	32 (28-38)	0.83

Abbreviation: PT, prothrombin time; APTT, activated partial thrombin time.

Data are presented as number (percentage), mean ± SD (normally distributed data) or median (interquartile range) (non-normally distributed data).

characteristics were not significantly different. The mean arterial blood pressure (MAP) change during the first post-SDH dialysis was not significantly different between the CPD and EHD groups (7 vs. 12, $p = 0.77$). Four patients in the CPD group and 4 patients in the EHD group received neurosurgery. One patient in the CPD group and 2 patient in the EHD group had enlargement of the hematoma, documented by a second brain CT. The 30-day mortality rate was 25% in the CPD and 14 % in the EHD group, which showed no statistically significant difference.

Five patients in the CPD group suffered from PD-related complications, including PD catheter dysfunction in two, dialysate leakage in four, and PD-related peritonitis in two patients. In the EHD group, three patients had non-fatal hypovolemic shock (systolic blood pressure < 90 mmHg, or mean arterial pressure < 60 mmHg without other etiology for shock). There was no episode of hyperkalemia or bleeding found in both groups. Dialysis-related complications were not associated with mortality.

The comparisons of baseline demographics and initial clinical characteristics in patients with and without 30-day mortality were listed in Table 3 (for the purpose of the present study, two patients received CVVHD were included in analysis here). All the variates were not significantly different between two groups, except that patients with 30-day mortality had higher rates of consciousness disturbance when presentation than those without (83% vs. 27%, $p = 0.02$). Neurosurgical interventions were done in nine (28%) patients, including procedures of Burr hole in three (9%) and craniotomy in six (19%) patients.

The details of neurosurgery and first dialysis therapy of patients with and without 30-day mortality were compared in Table 4. Lower score of Glasgow coma scale (GCS) (median [interquartile range]: 10 [7-12] vs. 15 [11-15], $p = 0.02$) and higher changes in absolute mean arterial pressure (MAP) (median [interquartile range]: 26.5 [10.5-46.0] vs. 7.5 [2.0-17.8], $p = 0.01$) were noted in patients with 30-day mortality. The unadjusted odds ratio (95% confidence interval, p-value) was 1.4 (1.03-1.9, 0.03) and

Table 4. Comparisons of neurosurgery and details of the first dialysis after SDH between patients with and without 30-day mortality

	With 30-day mortality (n = 6)	Without 30-day mortality (n = 26)	p-value
Neurosurgery	3 (50)	6 (23)	0.31
Time interval between SDH onset and neurosurgery, days	0.6 (0.2-4.0)*	4.3 (0.2-15)*	0.55
Time interval between SDH onset and first dialysis, days	6.0 (2-10.3)	3.5 (2-4.3)	0.24
GCS at first dialysis	10 (7-12)	15 (11-15)	0.02
Absolute MAP changes during first dialysis, mmHg	26.5 (10.5-46.0)	7.5 (2.0-17.8)	0.01
Labetalol or CCB treatment during first dialysis	2 (33)	6 (23)	0.63
Inotropic agent treatment during first dialysis	2 (33)	1 (4)	0.08

Abbreviation: SDH, subdural hemorrhage; GCS, Glasgow coma scale; MAP, Mean arterial pressure; CCB, calcium channel blocker.
*Median (range)
Other data are presented as number (percentage), mean ± SD (normally distributed data) or median (interquartile range) (non-normally distributed data).
MAP changes were recorded between the start and end of the first EHD, and of the first day of CPD.

2.6 (1.2-5.6, 0.02) for each point decrease of GCS score and each 10 mmHg absolute change of MAP, respectively.

Significant variables including consciousness disturbance, GCS and MAP in univariate analysis were included for multivariate analyses. GCS and MAP were transformed into category variables, using the median levels of 32 patients as the cut-offs (GCS < 12, MAP > 14). The results revealed that consciousness disturbance at presentation was an independent risk factor for 30-day mortality.

DISCUSSION

In this retrospective study, the 30-day mortality rate was 19% in our hospitalized SDH patients with ESRD requiring dialysis. Consciousness disturbance when presentation, lower GCS score and larger absolute changes of MAP during first dialysis therapy were noted in patients with 30-day mortality in univariate analysis, while consciousness disturbance is an independent risk factor for 30-day mortality.

The 30-day mortality rate of our cohort (19%), compared to 35-39% reported in two prior nationwide surveys of SDH in ESRD patients in Taiwan and in the United States[3,4]. A much lower 30-day mortality rate was also noted in our intracranial hemorrhage (ICH) patients with ESRD (28%) compared with 58-67% reported in the literature[13,14]. Different case-mix with more cases with mild disease severity should not be the explanation, since our center is a large tertiary referral hospital and admits more critical patients in the catchment area than hospitals in general. We speculate that different treatment strategy, e.g. the choice dialysis mode after SDH or ICH, may be a major determining factor for the lower risk of 30-day mortality in our patients.

For ESRD patients with acute brain injury, it is important to minimize cardiovascular instability during dialysis. For example, intradialytic hypotension will reduce MAP and cerebral perfusion pressure, leading to cerebral ischemia, increased intracranial pressure (ICP), or even death[5]. This concept was proved again in our cohort that larger changes of MAP when first dialysis therapy was significantly associated with 30-day mortality after SDH. In our center, we will shift intermittent HD to EDD, CPD, or CVVHD during the acute phase of brain injury in ESRD patients[8]. Those three dialysis modes may minimize the risk of intradialytic hypotension; however, it remains unclear that which one is the best[8,15]. PD has been suggested a preferred mode of renal replacement therapy in patients of ESRD who developed subdural hematoma on HD[11,12,16], because it involves dialysis support without the need of heparin. Continuous renal replacement therapy, such as CVVHD, were shown to cause fewer surges in ICP and more stability of cerebral perfusion

pressure than standard intermittent technique[17]. However, continuous dialysis therapies are generally used limited to the intensive care units setting and many HD patients with SDH are managed in the general wards. Thus, the dialysis prescription for these patients should be modified to minimized changes in hemodynamics during HD[5]. The modified HD protocol, such as EHD in our institution, can provide a relatively more stable hemodynamic state compared with traditional HD and a similar effect to PD and eliminate the need for anticoagulation (heparin free). The 30-day mortality rate were similar between the CPD and EHD groups, and both groups experienced complications. Although not associated with mortality, we revealed that the CPD group had a higher prevalence of complications related to PD catheter implantation or the dialysis itself, such as dialysate leakage, PD catheter dysfunction and peritonitis.

We also found that consciousness disturbance when presentation and lower GCS score during first dialysis therapy were both associated with 30-day mortality in univariate analysis, while consciousness disturbance at presentation was an independent risk for 30-day mortality in multivariate analysis. Those findings may be due to that impaired level of consciousness indicates the severity of SDH. Of note is that 22% of our patients did not have known history of head injury, while 47% of them were not diagnosed during the acute phase of SDH. High level of suspicion of consulting neurologists may be needed to diagnose SDH timely in ESRD patients who usually had multiple medical comorbidities, fragile underlying neurologic conditions, and higher bleeding tendency.

Our study had several limitations. First, the sample size was small that did not allow us to perform multivariate analysis. Second, we did not have data of ICP during dialysis changes in all our patients. Third, early palliative care would affect the outcome, but we did not record this information. Finally, this was a retrospective study with inherent potential for bias. Randomized clinical trial may be needed to determine the best dialysis mode for ESRD patients with SDH, as their ICH counterparts[15].

CONCLUSION

Among ESRD patients with SDH, those receiving EHD had a similar 30-day mortality rate to those receiving CPD. Absolute change of MAP during dialysis was associated with 30-day mortality after SDH in ESRD patients, and was a potential modifiable risk factor. An attempt to reduce the hemodynamic changes during HD might provide survival benefit for such patients.

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